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Attention Deficit Disorder: An Overview

Wiley C. Rasbury, PhD*

There have always been and probably always will be children who are hyperactive, impulsive, inattentive, and distractible. One of the first descriptions of such children was given by Dr. George Still (1) in 1902 in his lecture to the Royal Academy of Physicians in London. In the past 80 years interest in the study of children with these behavior characteristics has grown with respect to the etiology, management, and outcome of their condition.

More literature has been published on the group of behaviors now referred to as the attention deficit disorder (ADD) than on any other psychosocial problem in childhood. Approximately 31 publications were written on the ADD between 1957 and 1960, 2,000 between 1961 and 1976, 700 from 1977 to 1980 (2), and over 900 from 1981 through 1987. However, these statistics do not necessarily imply that the incidence of ADD is rising, and it is still impossible to make concrete epidemiological statements given the lack of agreement on a definition of the condition. However, substantial evidence shows that some children are significantly less attentive and more impulsive, distractible, and/or hyperactive than their peers and that such problems can interfere with their social, personal, and academic adjustment.

No precise definition of the ADD exists, and we do not know whether or not such childhood behavioral problems are more common today than in the past. Neither do we know exactly what causes such behaviors, nor how to best manage and/or treat these children, although Ross and Ross (2) and Weiss and Hechtman (3) have addressed these issues.

Definitions

While there is some consensus on the behaviors of this disorder, ambiguity still exists. As Conners and Wells (4) noted: "The symptoms appear to come from the same pot, but are sometimes stirred a bit differently. The range of symptoms involved seems for the most part to reflect a difference in emphasis rather than fundamental disagreement." In his review of over 200 studies on the ADD, Barkley (5) found that over 70% failed to use any objective or specifiable criteria for diagnosis. The definition by the American Psychiatric Association (APA) (6) is based on a specific set of behaviors which includes most of those of concern to researchers and clinicians (Table 1). Unfortunately, the APA definition, like most, lacks specificity regarding etiology, intensity of symptoms, pervasiveness, and numerous aspects of differential diagnosis and fails to clarify the definitional picture of the disorder. In clinical practice no universally agreed upon approach to diagnosis exists, but presumably most

clinicians rely on their experience with children to render a diagnosis based on age of onset of symptoms, intensity, pervasiveness, duration of specific behaviors, and exclusion of other psychopathological and nonpsychopathological childhood conditions that can cause such behaviors. While clinicians use a variety of methods to diagnose this disorder, generally the following criteria are useful: 1) history; 2) behavioral ratings on standardized forms completed by parents and teachers; 3) clinical observations; and 4) psychological test performance, especially on tasks of attention/concentration and learning (7). In the final analysis, an adequate or acceptable definition of the ADD will evolve slowly with continuing research. The operational definition offered by the APA, while not wholly acceptable, does attempt to reflect knowledge gained from research in this area and therefore may be considered as an acceptable operational guideline subject to ongoing refinement.

Prevalence

Prevalence estimates of the ADD vary from 1% to 10% with an average of 5%; estimates are significantly lower (typically 1% to 1.5%) when diagnosis is based on agreement of multiple sources such as teachers, parents, physicians, or other clinicians (8). Male:female estimates have ranged from 3:1 to 9:1, with a mean of 6:1. Cross-cultural studies utilizing teacher ratings alone have revealed estimates of 12% to 18% (8). These higher estimates, assuming they are valid, probably represent a heterogeneous group of children with respect to the factors causing their behavior, with a much smaller subset meeting a more rigorous definition of the disorder. Also, such data reflect the potential danger in basing a diagnosis of ADD solely on teacher ratings, especially if medication is recommended as the primary treatment approach. Of the children referred to our clinic, 50% to 60% have teacher ratings suggesting the ADD, but no more than 2% to 3% of these children receive the diagnosis.

Etiologic Speculations

Lack of etiologic knowledge before the 1970s has been noted in various reviews (9), but much progress has been made in the last decade in identifying possible causes of this disorder. While

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*Department of Pediatrics, Henry Ford Hospital.

Address correspondence to Dr. Rasbury, Department of Pediatrics, Henry Ford Hospital, 2799 W Grand Blvd, Detroit, MI 48202.

views have shifted away from unifactorial to multifactorial causes, no proposed explanation for the condition has been universally or even consensually accepted by researchers and clinicians. Furthermore, a number of thought-provoking articles have questioned the utility and validity of the attention deficit concept (10,11). Various areas implicated as possible explanations for the ADD, ranging from central nervous system damage to child-rearing practices, are discussed in the following sections.

Central nervous system damage

While injury or infection of the central nervous system can produce either a transient or chronic ADD, such damage accounts for no more than 5% of the children viewed as manifesting these behaviors. Numerous studies of children diagnosed with the disorder report that approximately 50% had "soft" neurological signs, but the etiologic relevance of these signs has been questioned. As Touwen and Sporrel (12) stated: "The attempt to explain disorders of complex behavior only on the basis of neurological minor signs testifies to an objectionable kind of reductionism."

Neuroanatomic

Recent reviews (13,14) revealed no less than 11 different (in some cases overlapping) theories of the neuroanatomic basis of the disorder. While none of these have gained significant empirical support, the most intriguing theories postulate a relationship between various subcortical brain structures and the frontal lobes.

Neurotransmitter

Much interest has been placed on the role of dopamine and, to a lesser extent, of norepinephrine on the mediation of the ADD. Interest in these sympathomimetic amines largely stems from use of the stimulants methylphenidate hydrochloride, dextroamphetamine sulfate, and pemoline which affect the release and reuptake of dopamine and norepinephrine (15) and help to reduce attention deficit behaviors. Several reviews have been published on the role of neurotransmitters in this condition (13,14,16,17). Zametkin and Rapoport (13,14) attempted to integrate neuroanatomic and neurotransmitter theories of attention deficit behavior. In general, as with neuroanatomic theories, there is no singularly accepted biochemical theory of the disorder. Furthermore, given the status of this research, it is inappropriate for clinicians to tell parents or the affected child that the attention deficit behavior is the result of a "chemical imbalance." Such statements are at best misleading and at worst counterproductive in the management of these patients.

Genetic

Genetic studies of families and adoptees provide some support for the possible genetic transmission of the ADD, but the research lacks sufficient scientific evidence (2). Researchers suspect that if the disorder is transmitted genetically, it is polygenic in nature. One of the more interesting theories of genetic transmission suggests that the disorder is part of a broader

Table 1
Diagnostic Criteria for the Attention Deficit Disorder*

Inattention (at least three of the following):†
Fails to finish what is started
Fails to listen
Easily distracted
Difficulty concentrating on schoolwork or other tasks requiring sustained attention
Difficulty remaining with a play activity
Impulsivity (at least three of the following):‡
Acts before thinking
Difficulty organizing work (not due to cognitive impairment)
Needs much supervision
Frequently calls out in class
Difficulty awaiting turn in games or group situations
Hyperactivity (at least two of the following):‡
Runs about or climbs excessively
Difficulty sitting still or fidgets excessively
Difficulty staying seated
Moves about excessively during sleep
Always "on the go" or acts as if "driven by a motor"
Onset before age 7
Duration at least six months
Not due to schizophrenia, affective disorder, or severe or profound mental retardation

*From American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3rd ed. Washington, DC: American Psychiatric Association, 1980.

†The number of symptoms specified is for children between ages 8 and 10, the peak age range for referral. In younger children, more severe forms of the symptoms and a greater number of symptoms are usually present. The opposite is true in older children.

constellation of disinhibitory conditions (eg, alcoholism, psychopathy, hysteria, antisocial behavior, and impulsive personality) (18).

Constitutional

Thomas and Chess (19,20) suggested infant temperament as a possible cause of attention deficit behavior. The numerous temperament styles delineated are assumed to reflect normal genetic variation in the expression of behavior. Theoretically, the behaviors delineating the ADD are reflective of constitutionally-based temperament patterns. The absence of any convincing pathophysiologic process in most children with the disorder makes the study of temperament patterns particularly interesting. Unfortunately, the methodological problems inherent in the study of temperament make it difficult, if not impossible, to make conclusive etiologic statements.

Food additives

In the 1970s, food additives were proposed as a major cause of the ADD in children. Conners (21) cast significant doubt on the validity of food additives as a relevant etiologic factor. At best, an extremely small percentage of children (3% or less) may manifest attention deficit behaviors secondary to problems with the metabolism of food additives. During the 1980s, studies were conducted on the role of sugar as a cause of these behaviors, but the results do not support such a relationship. Allergies, particularly food allergies, also have been suggested as a cause, but no convincing evidence has been reported (22).

Child-rearing patterns

Child-rearing approaches have been implicated as a cause of attention deficit behaviors, as well as parents who serve as potential primary role models for the imitation of such behaviors (2). Whether such experiences are sufficient to produce the ADD is unknown.

Social and emotional adjustment problems

Most clinicians realize that attention deficit behaviors can be present in children with primary emotional disturbances, acute stress, and/or conflict and tension within the family. However, it is unclear whether such problems can, by themselves, produce a child with the disorder. Attention deficit behaviors secondary to these psychosocial problems can lead to an erroneous diagnosis of the ADD and inappropriate management of the child's problems.

Academic failure and frustration

While attention deficit behaviors can interfere with optimal school performance, primary learning problems may also give rise to emotional lability, frustration, reduced motivation, and subsequently to attention deficit behaviors (5). In my opinion, many children with learning difficulties are misdiagnosed as having the ADD.

Treatment

Medication

Despite recent controversy over the use of stimulants in children with the ADD (23), stimulant medication is the most common treatment approach and probably the most effective as a symptom-suppression agent. Yet positive response to stimulant medication is not a confirmation of the diagnosis. Studies show that approximately seven of ten children, adolescents, or adults who are given stimulants will show improvement of the target behaviors for which stimulant medication is administered (ie, hyperactivity, inattention, distractibility, and impulsivity) (22,24). Beyond this base-rate responsiveness to stimulant medication, no single or multiple predictors can gauge responsiveness to stimulants. It was formerly thought that attention deficit behavior secondary to central nervous system damage could be reduced more effectively with antipsychotic medications than with stimulants, but no convincing evidence has been presented. Nor is there evidence that the use of stimulants eliminates (ie, cures the child of) attention deficit behaviors. Stimulants cannot teach the child how to behave in a socially, interpersonally, or academically adaptive manner, even though they may facilitate improved functioning in these areas. These types of skills must be learned.

The most commonly used stimulants are methylphenidate hydrochloride, dextroamphetamine sulfate, and pemoline. Methylphenidate hydrochloride and dextroamphetamine sulfate are essentially equal in their overall effectiveness and reported side effects, although dextroamphetamine sulfate is thought to suppress appetite more than methylphenidate hydrochloride. Pemoline is less effective than the other two but has similar side effects. Other psychotropic medications have been used, notably antipsychotics and antidepressants, but with less success

(14,25). Methylphenidate hydrochloride (5, 10, and 20 mg) and dextroamphetamine sulfate (5 mg) tablets are relatively quick acting (within 30 to 40 minutes) and of short duration (three to four hours). Methylphenidate hydrochloride also is available as a time-release preparation (20 mg slow release) which is effective for approximately eight hours, while the dextroamphetamine sulfate time-release preparation (5, 10, and 15 mg) is effective for approximately 12 hours. Pemoline (18.75, 37.50, and 75 mg) is administered once daily but requires two to three weeks of continuous administration before a therapeutic effect is noted. The development of tolerance to stimulant medication is rare (25). Preschool children (aged 3 to 5 years) need higher doses per kilogram of body weight than elementary school children (aged 6 to 12 years), and adolescents need lower doses per kilogram of body weight than elementary school children. In general, children under age 3 do not benefit from the use of stimulants. Approximately 50% of children aged 3 to 5 years who are given stimulants benefit from them, and approximately 75% of the children aged 6 years and older respond positively to medication (5). Although preschool children are less likely to benefit from stimulant medication and are more likely to exhibit side effects than older children (26), the use of stimulants in these children is widespread (8) and potentially beneficial in some cases (26,27). The most commonly recommended initial and maximum daily dosages of these medications are presented in Table 2.

Numerous texts provide comprehensive discussions of treatment side effects (5,8,28). The two most commonly reported side effects of stimulants are appetite suppression and insomnia. Safer and Allen (29) reported suppression of height and weight with chronic use of stimulants. However, subsequent research suggested that such suppression occurs most commonly in doses above 1 mg/kg/day with methylphenidate hydrochloride and at half this level with dextroamphetamine sulfate (30). Gross (31) noted that the suppressive effect is minimal over time and is often followed by a rebound effect when treatment is stopped. In a well-controlled study, McNutt and colleagues (32) found no evidence of suppression in height or weight over one year of treatment. Thus, as Campbell et al (33) noted, the controversy on the issue of height and weight suppression as a secondary feature to treatment with stimulants continues. Insomnia occurs most often when stimulants are given in the late afternoon. Hyperirritability and emotional lability, other potential side effects, are especially prominent as the medication wears off or as dosage is increased. These side effects can interfere significantly with the child's general behavioral, social, and academic performance. They can be misinterpreted as a need for more medication which, if given, only intensifies the negative effects. There is no evidence that stimulants are addictive or that they have long-term negative side effects as do the antipsychotic drugs (22). Psychotic reactions reportedly occur most often with dosages within the recommended therapeutic range, but these reactions have been infrequent (5,34). The physical and/or psychologic side effects of stimulant medication are minimal compared to those of all other psychotropic medications (8,35). There are potential indirect psychosocial effects of stimulant medication use. For example, children on medication might assume their behavior is solely a function of the "pill" and hence

feel no sense of personal responsibility for their behavior. Some children dislike taking the medication and feel they are "weird." The child's perceptions, as well as those of his/her parents and teachers, are important factors to consider when stimulant medication treatment is considered. Whalen and Henker (36) reviewed the psychosocial issues surrounding the use of stimulant medication with children.

While stimulant medication is a reasonable treatment approach, the conditions under which it should be recommended need to be identified. Some children with the ADD function well socially and academically. Should such children be given medication simply to make them less restless and more attentive? What should be done if parents refuse to give medication to their child, despite the appropriateness of the diagnosis of ADD? Do physicians, educators, and mental health professionals have the right to pressure parents to give their child the medication? Unfortunately, there is no singularly accepted or unambiguous set of guidelines to answer these questions. Guidelines published by the Committee on Children with Disabilities/Committee on Drugs (37) describe such issues as choice of medication, dosage, and the importance of alternative or adjunct treatment(s), but do not provide insight about whether or not to treat the child with stimulant medication.

Psychological behavior therapy

The efficacy of individual psychotherapy has been questioned for many childhood disorders (22), and no convincing evidence exists regarding the utility of such an approach with children who present with the ADD. Behavior therapy approaches have not fared much better (38). Educating parents and teachers on how to manage and help these children to function more appropriately may be far more important.

Outcome

In their review of the outcome of the ADD, Weiss and Hechtman (3) concluded that: 1) core symptoms (restlessness, attention difficulties, and impulsivity) tended to persist in adolescence and adulthood, although they may be manifested somewhat differently and less intensely; 2) a significant percentage (approximately 25%) of adolescents with a history of attention deficit behaviors manifested antisocial behavior; 3) both children and adolescents with a history of attention deficit behaviors had more problems with self-esteem, social skills, and impulsivity than control subjects; 4) while symptoms may continue into adulthood, most studies indicated that a substantial proportion of subjects (30% to 50%) displayed behavior indistinguishable from normal adults; and 5) approximately 20% to 25% of the adults had significant drug and/or alcohol problems and/or significant antisocial behavior.

Outcome is most likely related to the following factors: 1) intensity of symptoms, 2) social adaptability, 3) intelligence and academic achievement levels, 4) stability of the family, 5) the parents' ability to manage the child's attention deficit behaviors, 6) social/emotional adjustment of the parents, and 7) the ability of school personnel to manage the child's attention deficit behaviors.

Table 2
Medication for Children with the Attention Deficit Disorder*

Drug	Starting Dose	Maximum Dose
Methylphenidate hydrochloride	0.30 mg/kg	1.00 mg/kg
Dextroamphetamine sulfate	0.15 mg/kg	0.50 mg/kg
Pemoline	0.50 mg/kg	2.00 mg/kg

*From Donnelly M, Rapoport JL. Attention deficit disorders. In: Wiener JM, ed. *Diagnosis and psychopharmacology of childhood and adolescent disorders*. New York: John Wiley, 1985.

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